FILE 'HOME' ENTERED AT 12:51:29 ON 12 OCT 2001

=> file .receptor
COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 0.15 0.15

FILE 'MEDLINE' ENTERED AT 12:51:45 ON 12 OCT 2001

FILE 'LIFESCI' ENTERED AT 12:51:45 ON 12 OCT 2001
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=> s DIRS1
L1 1 DIRS1

=> d 1

L1 ANSWER 1 OF 1 MEDLINE
AN 2001527384 IN-PROCESS
DN 21448676 PubMed ID: 11564763
TI Cutting edge: STAT activation by IL-19, IL-20 and mda-7 through IL-20 receptor complexes of two types.
AU Dumoutier L; Leemans C; Lejeune D; Kotenko S V; Renauld J C
CS Ludwig Institute for Cancer Research, Brussels Branch, Avenue Hippocrate 74, B-1200 Brussels, Belgium.
NC RO1 AI51139 (NIAID)
SO JOURNAL OF IMMUNOLOGY, (2001 Oct 1) 167 (7) 3545-9.
Journal code: IFB; 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS IN-PROCESS; NONINDEXED; Abridged Index Medicus Journals; Priority Journals
ED Entered STN: 20011001
Last Updated on STN: 20011001

=> s interferon-like receptor
L2 0 INTERFERON-LIKE RECEPTOR

=> s interferon receptor-like and subunit
L3 0 INTERFERON RECEPTOR-LIKE AND SUBUNIT

=> s IFN receptor
L4 448 IFN RECEPTOR

=> d 4

L4 ANSWER 4 OF 448 MEDLINE
AN 2001397908 MEDLINE
DN 21342590 PubMed ID: 11448172

TI Type I IFN modulates the immune response induced by DNA vaccination to pseudorabies virus glycoprotein C.
AU Tudor D; Riffault S; Carrat C; Lefevre F; Bernoin M; Charley B
CS Institut National de la Recherche Agronomique (INRA), Unite de Virologie et d'Immunologie Moleculaires, Jouy-en-Josas, 78350, France.
SO VIROLOGY, (2001 Jul 20) 286 (1) 197-205.
Journal code: XEA; 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200108
ED Entered STN: 20010827
Last Updated on STN: 20010827
Entered Medline: 20010823

=> d 8

L4 ANSWER 8 OF 448 MEDLINE
AN 2001320061 MEDLINE
DN 21286452 PubMed ID: 11390453
TI Cloning and characterization of IL-22 binding protein, a natural antagonist of IL-10-related T cell-derived inducible factor/IL-22.
AU Dumoutier L; Lejeune D; Colau D; Renauld J C
CS Ludwig Institute for Cancer Research, Brussels Branch and the Experimental Medicine Unit, Christian de Duve Institute of Cellular Pathology, Universite de Louvain, Brussels, Belgium.
SO JOURNAL OF IMMUNOLOGY, (2001 Jun 15) 166 (12) 7090-5.
Journal code: IFB; 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
OS GENBANK-AJ297262
EM 200108
ED Entered STN: 20010827
Last Updated on STN: 20010827
Entered Medline: 20010823

=> d 20

L4 ANSWER 20 OF 448 MEDLINE
AN 2001033145 MEDLINE
DN 20501119 PubMed ID: 11046044
TI Receptor for activated C-kinase (RACK-1), a WD motif-containing protein, specifically associates with the human type I IFN receptor.
AU Croze E; Usacheva A; Asarnow D; Minshall R D; Perez H D; Colamonici O
CS Department of Immunology, Berlex Biosciences, Richmond CA 94804, USA.. ed_croze@berlex.com
NC CA55079 (NCI)
GM54709 (NIGMS)
SO JOURNAL OF IMMUNOLOGY, (2000 Nov 1) 165 (9) 5127-32.
Journal code: IFB. ISSN: 0022-1767.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200011
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001130

=> d 102

L4 ANSWER 102 OF 448 MEDLINE
AN 95349586 MEDLINE
DN 95349586 PubMed ID: 7623815
TI Ligand-induced association of the type I interferon receptor components.
AU Cohen B; Novick D; Barak S; Rubinstein M
CS Department of Molecular Genetics and Virology, Weizmann Institute of Science, Rehovot, Israel.
SO MOLECULAR AND CELLULAR BIOLOGY, (1995 Aug) 15 (8) 4208-14.
Journal code: NGY; 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199508
ED Entered STN: 19950911
Last Updated on STN: 19950911
Entered Medline: 19950829

=> s HKAEF92
L5 0 HKAEF92

=> s DIRS2
L6 0 DIRS2

=> s IFN RECEPTOR and subunit
L7 76 IFN RECEPTOR AND SUBUNIT

=> d 50

L7 ANSWER 50 OF 76 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2001:410208 BIOSIS
DN PREV200100410208
TI Structure-function study of the extracellular domain of the human type 1 interferon receptor (IFNAR)-1 subunit.
AU Kumaran, J.; Colamonici, O. R.; Fish, E. N. (1)
CS (1) Toronto General Research Institute, University Health Network, 67, College Street, Canadian Blood Services Bldg., Room 424, Toronto, ON, M5G 2M1: en.fish@utoronto.ca Canada
SO Journal of Interferon and Cytokine Research, (May, 2000) Vol. 20, No. 5, pp. 479-485. print.
ISSN: 1079-9907.
DT Article
LA English

SL English

=> d 76

L7 ANSWER 76 OF 76 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1994:10343 BIOSIS
DN PREV199497023343
TI Tumor suppressor activity of the cloned subunit of the type I IFN receptor.
AU Colamonici, O. R. (1); Porterfield, B.; Domanski, P.; Diaz, M. O.
CS (1) Dep. Pathol., Univ. Tenn., TN USA
SO Journal of Interferon Research, (1993) Vol. 13, No. SUPPL. I, pp. S51.
Meeting Info.: Annual Meeting of the ISICR (International Society for Interferon and Cytokine Research) on the Interferon System Tokyo, Japan October 24-28, 1993
ISSN: 0197-8357.
DT Conference
LA English

=> FIL STNGUIDE

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		8.91	9.06

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=> file .receptor

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		0.00	9.06

FILE 'MEDLINE' ENTERED AT 13:00:53 ON 12 OCT 2001

FILE 'LIFESCI' ENTERED AT 13:00:53 ON 12 OCT 2001
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=> logoff hold

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		1.88	10.94

SESSION WILL BE HELD FOR 60 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 13:01:49 ON 12 OCT 2001
Connection closed by remote host

L1 ANSWER 1 OF 1 MEDLINE
AN 2001527384 IN-PROCESS
DN 21448676 PubMed ID: 11564763
TI Cutting edge: STAT activation by IL-19, IL-20 and mda-7 through IL-20 receptor complexes of two types.
AU Dumoutier L; Leemans C; Lejeune D; Kotenko S V; Renauld J C
CS Ludwig Institute for Cancer Research, Brussels Branch, Avenue Hippocrate 74, B-1200 Brussels, Belgium.
NC RO1 AI51139 (NIAID)
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ED Entered STN: 20011001
Last Updated on STN: 20011001

STIC-ILL

Suppl. NO 10/1

367609

From: Wegert, Sandra
Sent: Friday, October 12, 2001 1:00 PM
To: STIC-ILL
Subject: ill 09265540

Please order the following article:

Thanks,
Sandy

Tumor suppressor activity of the cloned subunit of the type I IFN receptor.

Colamonici, O. R. (1); Porterfield, B.; Domanski, P.; Diaz, M. O.

Dep. Pathol., Univ. Tenn., TN USA

Journal of Interferon Research, (1993) Vol. 13, No. SUPPL. 1, pp. S51.

Meeting Info.: Annual Meeting of the ISICR (International Society for Interferon and Cytokine Research) on the Interferon System Tokyo, Japan October 24-28, 1993
ISSN: 0197-8357.

Sandra Wegert
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20

ANNUAL MEETING OF THE INTERNATIONAL SOCIETY FOR INTERFERON AND CYTOKINE RESEARCH

ISICR '93

PROGRAM AND ABSTRACT BOOK

W2-5

THE EXTRACELLULAR DOMAIN OF HUMAN INTERFERON ALPHA RECEPTOR: ISOLATION OF BIOLOGICALLY ACTIVE SOLUBLE AND INCLUSION BODY-DERIVED PROTEINS FROM *ESCHERICHIA COLI*:

N.Y. NGUYEN, R.D.C. HIRATA, D.E. LEVY, J.C. ENTERLINE AND M.H. HIRATA. Office of Therapeutic Research and Review, Center for Biologics Evaluation and Research, Food and Drug administration, Bethesda, MD, USA 20892

The gene coding for the extracellular domain of human interferon (IFN) alpha receptor (lacking the signal peptide) has been cloned in plasmid pGEX-2T at the EcoRI and BamHI sites as fusion with glutathione-S-transferase; expression was induced at either 30° or 37°C with 0.1 mM IPTG. The fusion protein, predominantly found in cytoplasmic inclusion bodies, was solubilized by lysis buffer (50 mM Tris pH 9, 1 mM EDTA, 1 mM PMSF, 1 mM DTT) containing 8 M urea and was refolded by dialysis in lysis buffer in the absence of urea; a small proportion (5% - 20%) of the fusion protein was isolated in soluble form by sequential cell disruption in lysis buffer in the presence of 2 mg/ml lysozyme, 0.45% CHAPS or 1% Triton X-100. Both soluble and urea-solubilized forms were purified by gel chromatography on Sepharose CL-2B followed by glutathione agarose affinity chromatography. Representative recovery (per liter of cell culture) of affinity purified soluble and urea-solubilized proteins were less than 250 ug and 750 ug respectively. Both forms of fusion proteins inhibited the antiviral and antiproliferative activities of 10 U of IFN alpha B or IFN alpha 2b. Receptor-ligand binding in solution using iodinated IFN alpha B (specific activity 1.4 uCi/ug) or IFN alpha 2b (specific activity 4.5 uCi/ug) indicated a K_d of approximately 1 x 10^{-9} M for the two forms of fusion proteins.

W2-6

TUMOR SUPPRESSOR ACTIVITY OF THE CLONED SUBUNIT OF THE TYPE I IFN RECEPTOR. O. R. Colamonici, B. Porterfield, P. Domanski, M. O. Diaz. Dept. Pathology, Univ. of Tennessee. Univ. of Chicago. October 1993

The Type I IFN receptor (IFN-R) has a multichain structure composed by at least 3 different subunits: the α subunit (110 kDa), the β subunit (100 kDa), and the cloned receptor subunit (75-80 kDa). We have previouly reported that expression of the cloned Type I IFN-R subunit in human IFNα-resistant and mouse IFNα-nonresponsive cells restore the antiviral response to several Type I IFNs in the absence of a concomitant increase of IFNα binding. We now report that expression of this receptor subunit in the human IFNα-resistant K-562 cell line (K-562/S.3 cells) dramatically decrease cell proliferation and the cell density at which growth arrest is commonly observed. Unlike K-562/R4.3 or K-562/R4.4 cells transfected with empty vector, injection of K-562/S.3 cells transfected with the cloned receptor subunit failed to form tumors in nude mice. These effects were observed in the absence of detectable Type I IFNs in the conditioning medium. Similar results were obtained when mouse L-929 cells were transfected with the cloned receptor subcloned into a retroviral vector. L-929 cells expressing the cloned receptor subunit were contact inhibited, had a prolonged doubling time, failed to form colonies in soft agar, and did not form tumors in nude mice. Furthermore, the sole expression of the cloned receptor subunit also induced a flat phenotype. These data demonstrate that the cloned subunit of the Type I IFN-R has tumor suppressor activity similar to that reported for two other components of the IFNα signal transduction pathway, p68 and IRF1. This suggests that the Type I IFN system is a tumor suppressor system whose alteration may favor the development of the malignant phenotype.